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ORIGINAL ARTICLES.

POST-DIPHTHERITIC PARALYSIS OF ACCOMMODATION.*

By F. L. Henderson, M.D.,
ST. LOUIS, MO.

January 15th, 1909, a boy 12 years old was brought to me complaining that he could not read. The history of the case revealed the fact that he had recovered from an attack of pharyngeal diphtheria two weeks before. The case was one of post-diphtheritic paralysis of accommodation which presented the usual symptoms as seen by probably all of you. The extraocular muscles were not involved. The pupil responded promptly to light and convergence. There was no other muscular paresis though the kneejerk and Achilles reflex were absent. Absence of kneejerk is noted in at least 33 per cent. of all cases with or without paralysis. The mother claimed, when questioned as to the voice, that it was more muffled than was natural, but I was unable to detect this symptom. Pulse and respiration were normal. The lad seemed to be making an unusually good recovery except for this one symptom—the paralysis of the ciliary muscle. I have seen this condition a few times and many of you have seen it often. Nevertheless, the peculiarity of this pathological manifestation impressed me as never before, and the following questions insisted upon an answer. First—Where is the lesion which causes this isolated paralysis? Second—How does the toxin of the Klebs-Loeffler bacillus cause this paralysis? Third—Why does the toxin of the Klebs-Loeffler bacillus para-

*Read before the St. Louis Ophthalmological Society, April 12th, 1909.

lyze the ciliary muscle more frequently than any other muscle in the body except the velum palati? The answers were not forthcoming from my inner consciousness but I expected to find them in any of our numerous text books. I consulted Nettleship, May, Posey, Carter, Theobald, Posey & Spiller, Fick, Norris and Oliver, Schmidt-Rimpler, Roosa, Tiffany, Juler, Berry, Thorington, Ball, Arlt, Jackson, Noyes, Fuchs, Parsons, De Schweinitz, Raney and Gowers, and found the usual and familiar symptoms —prognosis and treatment, but nothing as to the Where, How or Why. My curiosity was aroused when I discovered that even insignificant points such as these have not been thrashed out to a finality and I pursued the investigation. With your indulgence, I will give you what this investigation revealed, knowing well the subject is of small practical value. The nucleus of the third nerve in the floor of the aqueduct of Sylvius is composed of several groups of cells. The small cell nucleus which supplies those fibers of the third nerve which go to the ciliary muscle has been more or less definitely located. A branch of the third nerve is the only motor nerve to enter the ciliary ganglion. The short ciliary nerves proceed from the ciliary ganglion to the ciliary muscle and in this muscle form a plexus—the orbiculus gangliosus or ciliary plexus. The fibers from the ciliary plexus terminate in exceedingly delicate endings between the contractile cells. Contraction of the ciliary muscle relaxes the suspensory ligament and capsule of the lens. The circular fibers (Müller's muscle, or compressor lentis) are supposed to take more part in this process than the meridional fibers (Brücke's muscle, or tensor chorioidea). When the pressure of the capsule is thus relieved, the lens becomes more convex by an inherent elasticity. Increase in its convexity increases its focusing power. This ability to increase its focusing power constitutes the act of accommodation. If the power of accommodation is lost we will assume that it is due to a loss of the contractile power in the ciliary muscle. This loss of power must be due to some lesion of its motor nerve or inertia of the muscle itself. Does the toxin of the Klebs-Loeffler bacillus destroy the motor impulse in the terminal filaments—in the ciliary plexus—in the short ciliary nerves—in the ciliary ganglion—in the third nerve or at the origin of the third nerve in the brain; or do the muscle cells refuse to respond to the motor impulse? Donders, who first discovered paralysis of accommodation following diphtheria in 1860, supposed the lesion to be a hæm-

orrhage in the region of the oculo-motor nerve center (Schwenk). Faure had, however, three years earlier, reported six cases of "weakness of sight" following diphtheria. Swanzy says. "The lesion in these cases is probably a nuclear one, and the evidence points to miliary extravasation of blood in the floor of the fourth ventricle." If this disease, late in its period of convalescence, should so frequently cause an extravasation of blood, which pressed upon the small nucleus controlling accommodation it would be a very interesting phenomenon. For this extravasation to occur at identical points on each side of the aqueduct of Sylvius is still more interesting and yet this is what would have to happen to produce binocular paresis of accommodation. Only one case of unilateral paralysis of accommodation (with mydriasis) has been reported, that of Jefferson in 1873. For this hæmorrhage to limit itself to the accommodation nucleus and so rarely spread to the small adjacent nuclei of pupil contraction and convergence would be very remarkable. Luther C. Peter says in speaking of paralysis of individual muscles: "As a rule such localized palsies are followed by a more or less permanent injury to the part. Instances of this kind on record are, paralysis of the masseters, of the trapezii and of brachial monoplegia. These paralyses are due usually to acute nuclear degeneration or to small capillary hæmorrhages into the spinal cord." The paralysis of accommodation following diphtheria practically always recovers. Only one case (Mühsam's), I believe, has been reported in which it remained permanent. For pressure on the accommodation brain cells to always result in restoration of function—quite contrary to the action of pressure on nerve cells in other localities, gives us an array of improbabilities which renders the nuclear theory, in my judgment, untenable. For the motor impulse to be intercepted along the course of the third nerve, it would be necessary for the morbid element of the blood, left by the disease, to get into the nerve bundle and establish some form of inhibitory neuritis in those fibers going to the ciliary muscle without affecting the similar nerve structures with which it would come into equally as intimate contact. This seems to me to be highly improbable, to say the least. The next point of attack would be in the lenticular ganglion. I found somewhere the statement that Hughlings Jackson thinks the nerves passing through the ganglia are most attacked by post-diphtheritic paralysis. Stellwag says: "Where true paralysis of accommodation

occurs alone, or combined only with mydriasis, cerebral origin of the disease is not impossible, it is true, but is very rare. It almost always depends on an affection of the short root, or of the lenticular ganglion." The third nerve divides into two branches. The inferior branch divides into three more nerves. One of these three nerves goes to the inferior oblique muscle and from this branch to the inferior oblique is given off the short nerve which forms the inferior or motor root of the ciliary ganglion. I do not believe this little nerve has yet revealed any histological peculiarity which would render it more susceptible to the diphtheritic poison than other nerves in the body, and furthermore it is hard to understand why this poison should affect the accommodation fibers so frequently and the pupillary contraction fibers so rarely. This same line of reasoning applies to the ciliary ganglion and the short ciliary nerves up to and including the intra muscular plexus, the orbiculus gangliosus, in the ciliary muscle. Here the two sets of fibers separate; some going to the cells of the ciliary muscle and others to the iris. Völcker's theory, which is the one most generally accepted, places the lesion in the nerve endings in the affected muscle. It seems to me a good hypothesis to assume, that the influence of the diphtheria poison is exerted upon the accommodation fibers beyond the point of separation from those which go to the iris. In other words, it affects the fine filaments which proceed from the ciliary plexus to the termination at the contractile cells of the muscle. However, it is claimed the lesion is not in the nerves—that the motor impulse is properly conveyed to the muscle but that the muscle fibers have lost their power to respond to the impulse. Knies asserts that the toxin has a direct influence on the ciliary muscle. Leyden (1880) concluded that a trophic disease of both nerve and muscle in varying extent existed. Schwenk excludes lesion of the ciliary muscle by saying: "Since eserin produces the physiological effect on the ciliary muscle, the diphtheritic poison cannot affect this muscle." I must digress here to say there is a difference of opinion as to how eserin acts upon the ciliary muscle. Butler's Therapeutics (1902) attributes its influence to stimulation of the third nerve endings and Hare (1904) says it has no influence on motor nerves except to depress their peripheral endings when administered in toxic doses. The action of eserin he attributes to direct stimulation of the unstriped fibers of the ciliary muscle. If we accept Hare as correct, the

prompt response of the ciliary muscle to the action of eserin would be highly presumptive evidence that the muscle itself is not involved.

The question of how the toxin of diphtheria acts upon the terminal nerve filaments so as to temporarily suspend their functional activity is one which belongs to the bacterio-pathologist rather than the oculist and I would not go far into the subject if I could. Suter says in post-diphtheritic paralysis of accommodation "the diphtheritic toxin produces a peripheral ciliary neuritis." Paul Meyer (1881), Eichorst and Preisz found parenchymatous changes in the peripheral nerves. Cases of paralysis of the heart following diphtheria, Veronese, Klimoff and Thomas believe to be due to changes in the nerves of the heart muscle. Peter says in summing up the pathology of paralysis following diphtheria: "A more conservative view and the one generally accepted, is that the entire neuron, either motor or sensory, may be the seat of pathological changes, the peripheral neuritis usually predominating." All authorities practically agree that the lesion is caused by a toxin in the blood, the product of the diptheria bacillus and that the blood current is the distributor of this poison. It would seem then that the morbid blood established an inflammatory condition or a nutritive change in the fine terminal nerve filaments in the fibers of Müller's muscle which temporarily impairs their function. And when we say this, the smallest part of the question, How? is answered.

Posey and Spiller say 5 or 6 per cent. of all cases of diphtheria are followed by paralysis of accommodation. Brav puts the proportion at ten per cent. We must also consider that many cases are probably overlooked owing to the age of these patients.

And last, why is the ciliary muscle more frequently paralyzed than any other muscle in the body except the velum palati? Before taking up this question it is well to suggest that the reason why the velum palati is most frequently attacked may be due to its proximity to the seat of infection. This suggestion is more or less sustained by the case of Kussmaul which presented paralysis of the abdominal muscles following diphtheria of the navel. Helbron says it is not known why the toxin of diphtheria should so frequently attack the accommodation apparatus. He inclines to Bernheimer's explanation that the demand upon the accomodation during a period when the nutrition of the ciliary muscle

is low results in its failure to respond. He asserts that paralysis of accommodation is more frequent in hyperopes, whose ciliary muscles for obvious reasons are called upon for the greatest amount of effort. He cites the fact that out of 37 cases in his clinic, 3 were emmetropic, 1 slightly myopic, and the rest hypermetropic. Rest is second only to antitoxin as a prophylactic against all forms of post-diphtheritic paralysis. The most dreaded sequelæ of diphtheria and the most fatal are paralysis of the heart and of the diaphragm. Of 275 cases of post-diphtheritic paralysis reported by Meyer, 69 were cardiac (64 died) and 21 were diaphragmatic (11 died). The heart and diaphragm are involuntary muscles and their functions preclude rest. The ciliary muscle is also involuntary and restrained from activity with difficulty. Instead, however, of any effort being made to rest the ciliary muscle the probability is that the child is confined to its bed or room, to secure the muscular rest so necessary to a good recovery, and to amuse it, is provided with paper dolls to cut out, picture books to look at, or is given its school books to make up in a few days the weeks lost.

Bernheimer's theory that the ciliary muscle refuses to act because demands are made upon it at a time when its powers are diminished does not seem an adequate explanation of the great frequency of this isolated paralysis. This line of reasoning would apply to the whole muscular system and furthermore would apply to the ciliary muscle after many kinds of debilitating disease not followed by paralysis. Use of the muscle when in a weakened condition probably acts as an exciting cause but there must be a definite predisposing cause. Poisons have a predelection to paralysis of individual muscles or groups of muscles. I am not aware of any satisfactory explanation of why lead poison causes wrist drop, alcohol foot drop or why syphilis elects the posterior spinal roots, yet there is a reason which in all probability will be well understood in the future. We will exclude from consideration temporarily the part played by unrestrained exercise of the weakened muscles and assume that the predisposition to paralysis depends upon the amount of diphtheria toxin in the blood. It is highly probable that a muscle receiving a large amount of this morbid blood (the volume of the muscle being considered) will show a greater tendency to paralysis than one not so freely supplied. If this proposition contains any element of truth then the predisposition to post diphtheritic paraly-

sis of accommodation may be explained by the anatomical structure of the ciliary body. The circular fibers, the ones most potent in the accommodation act are relatively small in volume and lie close to the ciliary processes. In fact they are almost imbedded in these vascular glomeruli. Their position would seem to contribute to an intense saturation with the morbid element. And when we add to the free supply of toxin, the fact that the nerve filaments in Müller's muscle are very numerous, exceedingly fine and entirely denuded of isulation, we have sufficient explanation of the first or predisposing cause of paralysis of this little muscle. As stated before the involuntary muscles, probably as a result of their inability to rest, show great susceptibility to the diphtheria toxin, as in the case of heart and diaphragm. When we add to the difficulty of resting the ciliary muscle the fact that no effort is made to rest it, and on the contrary, for the entertainment of the convalescent, it is subjected to more than normal exercise, we have the second or exciting cause of its paralysis. The question will be asked why the pupillary contraction fibers are so rarely paralyzed when they, too, are imbedded in such a vascular body, and are also bathed in the aqueous humor in which the toxin is probably present (I say it is probably in the aqueous because Cobbett proved its presence in the urine). I would offer as an answer that though the same predisposing cause may be present the exciting cause is absent. The conditions surrounding the convalescent do not necessarily impose more than the usual amount of work on the pupillary fibers. Furthermore, I think it quite probable that the pupil contracting fibers in the physiological state perform their function with an expenditure of force which represents a smaller part of their total power than is the case with the ciliary muscle. For example, the asthenopic symptoms resulting from tired and exhausted ciliary muscles are constantly presented to us; but I am not aware that pain from over use of the circular fibers of the pupil has ever been suggested.

There seems to be some question as to the influence of antitoxin upon the subsequent development of paralysis. But the best opinion points to the conclusion that serum given early in the disease diminishes the amount and degree of post-diphtheritic paralysis (F. Ransom). Some think post-diphtheritic paralysis more common now than before the intro-

duction of anti-toxin, but this seeming discrepancy can easily be explained on the ground that the cases which now result in paralysis formerly all, or nearly all, died. Rainy's experiments on guinea pigs showed that paralysis was prevented by the use of serum in pigs injected with diphtheria toxin and that those in which no serum was injected had paralysis. Peter claims the tendency to paralysis is in direct proportion to the severity of the attack, but Groenouw says paralysis of extra-ocular muscles is apt to follow severe cases, whilst paralysis of accommodation may follow very light attacks. Brav says: "Post-diphtheritic paresis of accommodation is never a complete paralysis." Yet Schwenk says: "The total hypermetropia existing after diphtheritic paralysis of accommodation exceeds that which can be rendered manifest by any cycloplegic after the patient has recovered from the effects of the paralysis." Jefferson explains this greater hypermetropia of paralysis on the ground that the lens assumes a flatter curvature in prolonged paralysis of the ciliary muscle, than by a briefer paralysis induced by a cycloplegic.

In conclusion, first, the lesion seems to be in the terminal filaments in Müller's muscle. Second, this lesion seems to be an inflammatory neuritis. Third, the predisposing cause seems to be the excessive saturation of these non-insulated terminal filaments with the morbid blood, due to their arrangement in the vascular ciliary body. The exciting cause lies in the difficulty of resting this involuntary muscle and the fact that instead of attempting to rest it, it is subjected to unusual exercise. Finally, it seems wise when enforcing rest of all the other muscles of the body, to insure rest of the ciliary muscle by prohibiting all use of the eyes and keeping the child from school at least six weeks after convalescence begins.

LIGHTNING BURN OF THE EYE.*

By S. C. Ayres, M.D.,
CINCINNATI, OHIO.

Cases of injury of the eye from a stroke of lightning are sufficiently rare to justify me in reporting a case which came under my care in the summer of 1907. Injuries by lightning vary in severity from a slight œdema of the lids to optic neuritis. In fact any and all of the tissues which compose the eye may be involved, the cornea, lens, iris, retina, choroid, optic nerve, as well as the ocular muscles. With your permission I will give you a few selected cases of characteristic accidents of this kind.

Major Yarr, of the British Army in South Africa in 1900, reports two cases, in one of which there were several large retinal hæmorrhages in one eye, and in the other case there was detachment of the retina in both eyes.

Dr. Buller, of Montreal, in 1888, reported a case of severe injury from lightning stroke with loss of consciousness, in which there was in the left eye paralysis of the external rectus muscle, choroidal hæmorrhage and partial opacity of the lens.

Dr. Brixa (*Kl. Montasbl. f. Augen.,* 1900) reports the case of a telegraph operator struck while on duty. The following symptoms were noted: singeing of the eye lashes, œdema of the lids due to heat, congestion of the conjunctiva, especially around the cornea, iritis, and striae in the lens. Similar conditions obtained in both eyes. There was a para-central scotoma in both eyes and the fundus showed small black specks between the macula and optic disc.

Dr. Gonin (*Annales d' Oculistique,* 1904) reports two interesting cases which came under his personal observation. On the morning following an electric storm, the patient found himself almost blind in the right eye, the pupil was dilated, the optic disc pale, and the retinal arteries moderately contracted. Two years later V=1/12.

In the second case the patient was rendered unconscious for fifteen minutes. After recovering he found he was blind in the left eye and barely able to tell light from darkness in the right. His face and eyelids were much swollen, the pupils contracted and the irides reacted sluggishly. There was a diffuse corneal haze which disappeared and at the same time a stellate opacity

*Read before the Academy of Medicine of Cincinnati April 5.

of the right lens made its appearance. A month later the lenticular lesions remained unchanged. R. E. V—1/2; L. E. V—1/6. In addition to these cases Dr. Gonin collected twenty-one similar cases where lesions of the crystaline lens followed exposure to lightning. In eight cases affections of the optic nerves were found, a few cases of probable retinal hæmorrhage and one of choroidal rupture with retinal detachment. In his judgment these lightning lesions are due to the mechanical action of the current on the integrity of the histologic elements as well as the electro-chemical phenomena which are developed by the current. The cause of cataract after lightning has been explained on various grounds, the development of heat, the severity of the concussion and to the chemical effect of the electricity. Experiments were made by Hess of Würzburg on animal eyes, and his conclusion is that cataract is due to death of the epithelial cells of the lens capsule.

In the *London Lancet* of January 2, appears a very interesting article on freaks of lightning. It relates how eight men who were taking their mid-day meal under an oak tree were struck by lightning and instantaneous rigor mortis set in, and they were all found in the position they were in when struck—one had a glass in his hand, another was about to take a bite from a piece of bread which he held in his hand, and a third had his hand on a plate. At another place a woman was picking a poppy and was found standing with the flower in her hand. Let me say incidentally that several cases of instantaneous rigor mortis were reported during the Civil War where men were found dead but standing in the position they had when shot. These were usually penetrating wounds of the brain.

The *Lancet* reports cases where persons struck by lightning were stripped of their clothing, where the bones of men and animals were broken and crushed, where the hair, beard and eyebrows were burned off, and where both hearing and sight were destroyed.

The case I have to report is as follows: The accident occurred July 7, 1907. Mr. L., my patient, was standing near the gunnel of a boat in the Little Miami river, and a friend was near him. The bolt of lightning struck the boat with great force stunning Mr. L. very severely, but not rendering him unconscious. His friend was knocked from the boat into the water. His injuries were so severe that he died the next day. I saw Mr. L. early in the morning after the injury and found him suffering severely

from pain in the eye and eyelids. The latter were red, swollen and œdematous. The upper half of the cornea was hazy, resembling the appearance of an eye with acute glaucoma. There was no iritis and no abnormal tension. Cold applications were ordered and cocaine used freely to relieve the pain. Two days later there was a deep injection of the scleral vessels; a solution of adrenalin was used in conjunction with the cocaine which gave some relief. Three days later leeches were applied as the pain persisted; they had an excellent effect and relieved the deep seated pain which the cocaine and hypnotics failed to do. The swelling of the lids subsided and the cornea cleared up. A month from the time of the injury the vision was practically perfect with ametropia corrected.

OCULAR COMPLICATIONS OF SCARLET FEVER AND DIPHTHERIA.

J. H. Parsons (*Practitioner,* Jan., 1909) says that ocular complications of scarlet fever are not frequent. Among those which occasionally do occur are: conjunctivitis, which may destroy the eye; abscess and gangrene of the lids; complications secondary to scarlatinal nephritis, of which uræmic amaurosis is most frequent; retinitis, embolism, optic neuritis, orbital cellulitis, which are of very rare occurrence. Parsons believes membranous conjunctivitis to be the most frequent ocular complication of diphtheria. As the xerosis bacillus cannot be differentiated morphologically from the Klebs-Löffler bacillus, he suggests that membranous conjunctivitis may be caused by the former bacillus. Associated with the diphtheritic bacillus in this disease are usually found streptococci, staphylococci and other pathogenic microorganisms. Gangrene of the lids; diphtheria of the lacrimal sac, dacryoadenitis, orbital abscess, and optic neuritis may rarely occur. Post diphtheritic paralysis may involve the ciliary muscle and the iris and hence it is highly important that children who have had diphtheria should have both their distant and near vision tested before having a mydriatic use.

SCLERITIS WITH PERFORATION AND FORMATION OF A CONJUNCTIVAL "CYST."*

By J. W. Charles, M.D.,
ST. LOUIS, MO.

The recent report by Dr. W. M. Carling before the Colorado Ophthalmological Society of a case of "Rupture of the sclera", which was published in the *Ophthalmic Record,* March, 1909, resembles so strongly the history of a case at the Female Hospital in 1900 that it seems proper to add the latter to the literature of such cases.

Dr. Carling's case was that of a 24 year-old woman who gave "a history of inflammation of the right eye and neuralgic pain for four weeks previous" and "rheumatism prior to any ocular disturbance."—Iritis, complete posterior synechia.—Anterior chamber half-filled with blood.—Ciliary tenderness, bulging of the sclera on the temporal side.—Light perception.—Rupture three days later, with escape of vitreous. The rupture soon closed, but the eye softened and vision was limited to counting fingers.

My case was as follows: Ann W., colored, 60 years old, was admitted to the hospital giving a history of rheumatic pains with eye-pains and a lump above and to the inner side of the cornea. On January 10th, I saw the patient for the first time, and, with my limited experience in such affections, the condition was quite puzzling. There was an ulcer of the cornea in the upper inner quadrant with slight hypopyon.—Iritis.—Ciliary tenderness marked. Patient constantly suffered great pain.—Vision=digits at one foot.—Occupying almost the entire upper inner quadrant of the sclera, was what appeared to be a conjunctival cyst, with a perfectly black base, and filled with a very muddy gray fluid,—so cloudy that the base could not be definitely bounded. The very tentative diagnosis of a "melanotic cyst" was made and argent. nitrat. gr. i to ℥i and oft-repeated applications of atropine and cocaine were prescribed. I feel sure that small doses of calomel for a few days and then bichloride of mercury gr. 1/12th t.i.d. were also given, but my notes do not mention it.

Upon seeing the patient three days later, I found the ulcer much cleaner, the hypopyon almost gone and vision=digits at

*Read before the Ophthalmic Section of the St. Louis Medical Society, April 14, 1909.

eight feet. There was medium dilatation of the pupil and the "cyst" had entirely disappeared, leaving a large round puncture in the sclera over the ciliary region and back of it fully 3 mm. in diameter (or about the size of a small lead pencil) as if the sclera had been cleanly trephined. The surrounding sclera was thinned. Of course the "melanotic" appearance of the base of the "cyst" was due to the diffuse refraction from the uveal pigment through the cloudy fluid. Treatment was not changed.

On January 18th, I found that treatment had been discontinued for over twenty-four hours because the patient had had no more pain and she had been overlooked by the nurse(!) There was again exclusion of the pupil and the cyst was again present. Treatment was resumed, the pupil dilated and the "cyst" vanished. A few days later, in spite of our protest, the patient left the hospital because she was in no pain. Her vision when last seen was digits at 10 feet.

On one of my visits, she told us that she did not know of the presence of the lump until she had visited our clinic at the O'Fallon Dispensary in August, 1899. Her record at the clinic was short. Ann W., colored, gumma or neoplasm above and to the nasal side of the cornea O. D. Atropine and cocain instilled and the patient probably left without prescriptions since no record was made of further treatment or visits.

There was absolutely no history of injury.

We had then to do with a perforation of the sclera following inflammatory adhesions of the conjunctiva to the sclera, so that, when the vitreous escaped there was no sudden pain nor extensive loss of vitreous. When the pupil had become excluded by the iritis and the normal exit of fluid through the aqueous was obstructed, a subconjunctival reservoir was at hand which prevented further increase of tension. As to the cause, it was undoubtedly a scleritis originating in the ciliary body and chorioid accompanied by necrosis of scleral fibers. Modern pathology tells us that all deep inflammations of the sclera begin in the uvea.

The histologic findings in superficial episcleritis published by Dr. Alt (in the *American Journal of Ophthalmology*, April, 1903) explain the inflammatory adhesions of the conjunctiva to the sclera: "The conjunctival epithelium is thickened. Underneath it lie the greatly congested conjunctival bloodvessels surrounded by varying masses of round cells. The conjunctival

and episcleral tissue is somewhat œdematous and its elements are pressed apart. In it are seen a number of evidently enlarged lymph-vessels, and their number appears greater than in the norm. The episcleral bloodvessels are enormously engorged, so that they form almost a continuous layer, *the small interstices of which are filled with round cells. The round cell infiltration reaches also into the superficial layers of the sclerotic*" (all italics mine) . . . "The normal elements of the tissues did not seem to be materially altered in any of these cases."

In regard to deeper scleritis, he says, "While in these cases the episcleral tissue is always involved in the process of inflammation, it is especially the scleral tissue which suffers. There is usually a very considerable infiltration of round cells in the superficial layers of the sclerotic fibers which quite frequently assumes the shape of tubercle-like round and oblong nodular formations. *The pressure leads to hyaline degeneration or necrosis of the scleral fibers.* Friedland also found some giant cells. The pressure of the increasing number of round cells may also lead to necrosis in the round cell accumulations themselves, so that they lose their cellular characteristics and form a more uniform semi-hyaline mass. Later on, when the inflammation has subsided, the sclera is thin, there are few cells visible in it, the bloodvessels are obliterated and the corresponding part of the uveal tract is also atrophic and attached to the sclerotic."

"When the scleritis is still more virulent, we find also the inner layers of the sclerotic and the corresponding part of the uveal tract infiltrated with round cells, so that the boundary line between the two membranes cannot be distinguished. Between the infiltrated superficial and deep layers of the sclerotic there is usually a middle part which seems but little affected. *In other cases the infiltration concerns all the layers of the sclerotic.* All episcleral and conjunctival bloodvessels are gorged with blood and the lymphvessels appear to be much more numerous and larger than in the norm. *Necrosis of the scleral fibers* and of the infiltrating cells is usually seen in these cases." The result was atrophy with staphyloma or as in phthisis bulbi "hypertrophy."

Although giant cells had been found no tubercle bacilli were demonstrated nor could syphilis explain the histological findings. He did not believe that syphilis or rheumatism was the cause in the majority of cases. He also considered the deeper form secondary to uveal disease.

Since the paper of De Schweinitz on the coincidence of diseases of the cornea, sclera and uveal tract with autointoxication (*A. M. A.*, 1908) and the tuberculin tests of Verhoeff (*Boston Medical and Surgical Journal*, March 14th, 1907) our ideas on the ætiology of scleritis have undergone considerable change. Verhoeff used tuberculin injections in 13 cases (all women, average age 26 years), obtaining a positive general reaction in all, and a local reaction in 9. Excised nodules from 4 cases contained epitheloid cells and giant cells—no tubercule bacilli. The treatment of 4 cases with old tuberculin was very effective. Interstitial keratitis was present in all cases. In 3 cases there was a sluggish iritis. (*Ophthalmic Year Book.*)

A CASE OF BLUEISH DISCOLORATION OF THE CONJUNCTIVA FROM AN INDELIBLE PENCIL.

Aaron Brav (*N. Y. Med. Jour.*, Jan. 2, 1909) reports the case of a bookkeeper who, while sharpening an indelible pencil had a small piece of the lead get into the conjunctival sac, promptly discoloring the eye. When Brav saw the case shortly afterward he found a slight laceration of the conjunctiva of the lower lid, and the conjunctiva of this lid and of the globe as high as the lower edge of the cornea was a dark purple color. The patient being very anxious to know whether this would disappear, inquiry was made of a reliable chemist as to the chemical nature of the indelible lead and its possible reaction with the tissues of the body. The information was obtained that the indelible pencil contained the aniline dyes and that no special reaction occurred between it and the body tissues whereby insoluble salts were formed. A wash of sodium biborate was given the patient and on her return the following morning no trace of the stain could be found, the patient stating that the eye was clear by six p.m. the previous day. The author reported the case for the benefit of other physicians who might in the future be called to treat such cases.

MEDICAL SOCIETIES.

OPHTHALMIC SECTION
ST. LOUIS MEDICAL SOCIETY.

Meeting of January 13th, 1909.
Dr. A. E. Ewing, presiding.

A Case of Suppression of the Secretion of the Aqueous Humor. —By Dr. W. A. Shoemaker.

J. M. C., aged 42, consulted me on December 14, 1908, giving the following history. Was chopping wood one week ago, when a piece struck him in the right eye, slightly wounding the edge of the lower lid and at once completely blinding him. He is positive that he could not distinguish light from darkness with this eye for several hours, after which time vision began to return and by evening, the accident having occurred in the morning, he could distinguish large objects.

His vision remained blurred and two days after the accident he applied at one of the clinics for treatment. He took treatment for the succeeding five days, and finding his vision no better, consulted me on the morning of December 14, when I found the eye in the following condition:

V.=5/200. Ciliary and conjunctival injection, some photophobia, lacrimation and pain. Pupil about 4 mm. in diameter, anterior chamber extremely shallow and the tension about —2. As the iris was inflamed, atropine was used to dilate the pupil. Some posterior synechiae were found which disappeared by the next day. The ophthalmoscope revealed deposits on Descemet's membrane, and a hazy vitreous.

Bichloride of mercury in 1-16 gr. doses was given internally, three times daily. The next day, December 15, the pupil was dilated ad maximum, there was less ciliary injection, no pain and the vision was 13/200. Tension about —2. On December 16, V.=13/70. December 26, ciliary and conjunctival injection gone, vitreous much less hazy, and vision 13/50; W.—.50 D. S. —.50 D. C., axis 75=17/20. January 5, 1909. V.=13/20. Vitreous clear. Anterior chamber shallow as at first. T. —1. January 13, eye quiet, V.=13/30. W. —.50 D. S. =17/15.

Reads J. 1 at 14 feet W. + 1.50 D. S. T. —1, Ant. chamber shallow as at first visit. V. O. S. =17/15 W. + 1 D. S.=17/15, J. 1 at 13 feet. The temporary, complete loss of vision in this case was probably due to commotio retinæ which was caused, as it usually is, by the eye being struck. The shallow anterior chamber accompanied by minus tension can, it seems to me, be accounted for only on the supposition that there is a suppression of the secretion of the aqueous humor. This is a condition that, so far as I know, is quite rare.

A cursory examination of the available literature reveals only one case, which was reported by D. C. Lloyd-Owen in the *Ophthalmoscope,* August 1, 1906. A professional man, aged 40, who always enjoyed good health and good vision, complained of having blurred vision in his right eye, with a slight soreness, for one week. Owen found the anterior chamber very shallow, pupil active and normal in size, some tenderness over the equatorial region, and tension —2.

This condition lasted for one month when his sight suddenly became normal for a few hours. One week later, his vision again became normal, at which time the author found the anterior chamber and the tension normal. Four days later he had another relapse of three days, after which the eye remained normal for fourteen days, followed by a relapse of three days, a recovery for a month, two days relapse and then permanent recovery.

Retinal Degeneration Simulating Retinitis Circinata.—By Dr. John Green, Jr.

Mrs. M. G., married, aged 65 years, Russian, came to my service at the Protestant Hospital Dispensary, December 26, 1908. She stated that the vision of left eye had been defective for some time. On December 25, the vision of the right eye suddenly grew dim. R. E. with plus sph. V. 20/1000, L. E. with plus 1 sph. V. 20/50. The opthalmoscope revealed in the right eye retinal degeneration and hæmorrhages. The patient was put in the hospital, and a careful physical examination by Dr. M. L. Warfield, revealed nothing in the general system to account for the retinal degeneration.

Urinalysis showed absence of alubumin, sugar, blood and bile. No increase in indican. Microscopically, epithelium and pus cells, no casts. Under potassium iodide the hæmorrhages have

partially resorbed, but otherwise the fundus picture is much the same as at first examination. I present the case mainly to elicit suggestions as to how it should be classified. Its general appearance is suggestive of retinitis circinata.

DISCUSSION.

Dr. F. L. Henderson said that Dr. Green had referred to the possibility of his case being retinitis circinata. Dr. Henderson had had occasion to see two cases of retinitis circinata in the last two years. When he saw the first one he had looked up the picture of the disease as presented in Dr. de Schweinitz's report, and found it almost identical with the picture of the case he had in hand. He thought that Dr. Green's case showed very little resemblance to retinitis circinata. The arrangement if the white deposits was not sufficiently circular to give it that name. As he understood retinitis circinata, it was a name for a form of retinitis distinguished by its peculiar anatomical arrangement. These exudates, or whatever the peculiar tissue is, are arranged in circular form and unless so arranged, he did not believe it should be called a retinitis circinata.

Successful Removal of a Fragment of Steel Embedded in the Crystalline Lens, Followed by Linear Extraction.—By Dr. W. H. Luedde.

J. C., age 40 years, formerly base ball player, now carpenter, working for the American Car and Foundry Co., presented himself at the O'Fallon Dispensary, December 23, 1908, for treatment for his left eye.

His statement was that four days before, while trying to loosen a steel chisel, which he struck on the sharp end with a hammer, a small piece broke off and lodged in his left eye.

When I first saw him, the lens of the left eye was found clouded, a faint red reflex being present at the periphery, photophobia and circum-corneal injection were marked, pupil small, and iris adherent to the anterior capsule of the swollen lens.

Further examination showed what appeared to be the scar of a perforating wound in the center of the cornea. Immediately behind this, lying with its point forward, in the antero-posterior axis of the lens, was a small sliver of metallic substance, probably steel. The removal of this was accomplished by the aid of Dr. Charles with his giant magnet. Eleven days later a linear

extraction was done to remove the swollen lens substance, which was causing pronounced secondary glaucoma.

There have been no unpleasant complications and the vision now is, fingers at two feet, which will no doubt improve as the soft lens substance, which still is present in the pupil, will be absorbed.

DISCUSSION.

Dr. J. E. Jennings had had a case recently which might be of interest. A young man, about 20 years old had had a hammer and hatchet in his hand tapping them together. While doing that, something struck him in the right eye. He went to drug store and was given a lotion which he applied to the eye. His eye cleared up and did not hurt him at all. He only consulted Dr. Jennings on account of the dimness of vision in that eye.

Examination showed a localized opacity in the upper part of the lens; the iris seemed to be attached at the point, and there was visible a very small wound in the cornea. The localized opacity in the lens seemed to point to the presence of a foreign body, so the giant magnet was used. As soon as the current was turned on, the lens was drawn forward. The doctor thought he could detect the end of a piece of steel. He made a small incision, and introducing an iris forceps, picked it out.

OPHTHALMOLOGICAL SOCIETY OF THE UNITED KINGDOM.

Meeting, Thursday, January 28th, 1909.

The President, Mr. Marcus Gunn, in the chair.

CARD SPECIMENS.

? Tubercular Mass in the Choroid.—Mr. A. W. Ormond.

L. H., female, aged 39, had had defective sight in the left eye for 3 months. R. V.=6/6; L. V.=6/24. In the left fundus situated below and to the outer side of the optic disc was a solid soft-looking mass pushing the retina forwards, the top of which was seen with a +7; the margins were ill defined, fringed with hæmorrhages, and there was no pigmentation. On the assumption that the growth was of a tubercular nature .005 c.c. of T.O.A. was injected, and in 28 hours a reaction had occurred,

the temperature rising to 99.4°; 4 days later a dose twice the strength of the first was administered and the temperature rose in 12 hours to 103.8°. Koch's Tuberculin (T.R.), .0001 mg., was given at intervals of 14 days; after 3 months treatment the swelling had not diminished in height though its boundaries had contracted.

Peculiar Opacity of the Cornea—Mr. R. W. Doyne.

A. J. B., aged 64, male, was first seen in June, 1905. Opacities were seen both in the corneæ and the lenses; the patient had suffered from diabetes for the last 2 years. There was some myopic astigmatism which had been corrected with glasses; and whereas in 1905 the vision in each eye with correction was 6/12 pt., it had deteriorated to 6/18 in the right and 6/36 in the left by the end of January, 1909. In 1896 it was known definitely that the corneal opacities did not exist.

The condition shows a haziness of the cornea occupying the whole of the central area extending almost up to the limbus and sharply defined at the periphery.

In the discussion on this case, it was generally thought to be of the nature of a degeneration, similar to transverse band opacity of the cornea only of wider extent.

PAPERS.

Four Generations of Lamellar Cataracts.—Mr. N. Bishop Harman.

Mr. Bishop Harman showed a pedigree of a family of five generations, four of which presented a marked inheritance of congenital cataracts. Seventeen childships were traced comprising 63 persons; 8 died in infancy, and of the remaining 55 it was known that 19 had congenital cataract. In no case had there been any in-breeding, or marriage with a person from a family similarly affected. The type of cataract was for the most part central opacities, of varying size, some were minute Y-, X-, or star-shaped, while others showed definite lamellar formation. In some cases the cataracts were of long standing and no evidence could be found that they progressed beyond the stage at which they were first seen. Probably the number of cases of cataract found does not represent the true total, for in one childship of seven members, amongst whom only one case of cataract was admitted, examination of the seven showed that four had lenticular opacities.

The inheritance was strictly from those who had cataract, in no case was cataract found in a child where the parents were free from the defect. When the defect had disappeared from a branch of the family it did not tend to reappear.

The teeth usually were good, and no other development errors were found.

An Unusually Rapid Development of Complete Cataract in a Boy.—Mr. N. Bishop Harman.

The case described was that of a boy aged 9¾ years, whose mother stated that nine months ago the sight was good in both eyes, after which it got suddenly worse, so that when he came under observation he was practically blind. Both lenses were found to be completely cataractous, and no cause whatever, either in the boy's own history or in the family history, could be found to account for the condition. After curette evacuation the vision obtained was 6/6 in each eye.

Note on the Colors of Benham's Top.—Mr. A. S. Percival.

Mr. Percival considered that the phenomena observed with this top support the theory of Young-Helmholtz in regard to color vision. He quoted from a paper by Mr. G. N. Stewart in the Proceedings of the Royal Society of Edinburgh for 1888, in which experiments were described in relation to Talbot's law, and he finds that there is no appreciable departure from this law even where the stimulus lasts only 1/8000000 of a second; and he also shows that where the stimulus acts a long time there is an excitation of all colors, giving the sensation of white; if on the other hand the stimulus acts for a shorter time then violet preponderates, and again if for a very short time the red color is more prominent. In connection with the colors seen in Benham's Top Mr. Percival showed that the phenomenon depends on the irradiation of the surrounding white area over the edges of the black line, and the actual color produced is dependent on the rate and direction of rotation.

Note on Some Rhythmic Oscillations of the Pupil.—Mr. A. S. Percival.

Rhythmic contraction and dilatation of the pupils is well known as a phenomenon in many diseases; and Mr. Percival

compared this with the oscillatory discharge from a Leyden jar. It is found that if the discharge is made through a conductor of high resistance such as a recently dried linen thread, it simply dies away, the current at first increasing in strength and then gradually fading. If, however, the discharge is made through a coil of wire of lower resistance, the effect is wholly different, for then the discharge consists of a number of excessively rapid oscillations. If we may assume that nerve cells and axis cylinders represent the Leyden jar and conducting wire then we may surmise that the nerve fibres exhibit the same phenomenon of induction as the wire. In retrobulbar neuritis, for example, owing to nerve atrophy, the resistance decreases because the fibers are more closely in contact and the oscillatory movements take place. The same explanation can be applied to the condition of the pupils found in disseminated sclerosis.

A Case of Retinal Exudation with Extreme Distension of Vessels, and perhaps Arterio-venous Anastomosis.—Mr. D. J. Wood.

This paper was accompanied by a colored drawing illustrating the condition of the fundus; and in the course of his remarks the author alluded to similar cases described in the *Transactions of the Ophthalmological Society,* Vol. xii, p. 143, 1902, and Vol. xxv, page 96, 1905.

The patient, a female, was first seen in 1902, when two large bloodvessels were seen running up to a white patch in the upper and outer part of the right fundus. In November, 1907, when the patient was 50 years of age, the right eye failed suddenly, only large objects being visible. At first, owing to difuse opacity in the vitreous, no details of the fundus could be made out, but later on a white area was seen upwards and outwards from the disc with two enormously dilated vessels running up to it; their color was intermediate between arteries and veins, but from the left trunk a vessel sprang which was arterial in color, while another vessel, evidently venous, was given off from the opposite trunk. The question arose as to whether the condition was sarcomatous, but judging by its similarity to the other cases described, it was decided not to excise the eye but to watch for a time its subsequent course.

Mr. Coats referred to similar cases which had come under his notice, and were embodied in a paper appearing in the last number of the Royal London Hospital reports.

Meeting of February 11th, 1909.

Mr. Nettleship in the chair.

1. *Finished Models of the Diaphragm Test.*—Mr. N. Bishop Harman.

The models for the employment of this test, which were described at the meeting of the Society in December, 1908, were now shown in their completed state.

2. *Tobacco Amblyopia; a Substitute for Smoking.*—Mr. N. Bishop Harman.

The difficulty which many smokers have in giving up tobacco Mr. Harman found was alleviated by chewing quassia chips; and he had consequently instructed Fuller's to prepare it in the form of a chewing gum containing 1 in 20 of quassia extract.

3. *A Case of probable lack of Separation of M. Levator Palpebrae Superioris and M. Rectus Superior; Unilateral.*—Mr. N. Bishop Harman.

D. S., a healthy girl, aged 10, said that her eyes had always "appeared strange." There was a history of not being able to open the right eye for the three days after birth. No instruments were used at the child's birth. The child could fix with either eye but preferred the left; and the appearances differed according to which eye was for the moment the fixing one. If the right eye fixed, the left upper lid dropped 3 mm. and the eye turned slightly down and in. If on the other hand the left eye fixed, the right turned up and in. During concomitant movements, those of the left were perfect and full, while the right failed to follow the left in extreme elevation.

Mr. Harman considered that the only reasonable explanation was that there had been a failure in the separation of the original muscular mass which produces the levator palpebrae superioris and superior rectus; such incomplete separation is common in lower vertebrates.

Obstruction of Cilio-Retinal Artery.—Mr. A. Levy.

A colored drawing accompanied this case. The patient, a girl aged 22, complained that 6 days previously the vision in the right eye suddenly failed. During a previous visit to Moorfields on

account of squint her vision had been recorded as 6/6; it was at the present time 6/60. Stretching for 3 or 4 disc-diameters outwards was a broad patch of exudation, coinciding with the distribution of a vessel beginning at the outer margin of the disc. There was some evidence of arterio-sclerosis in the other retinal vessels.

A Case of Anirida.—Mr. J. F. Cunningham.

The case was that of a little girl aged 10, and the family history had been traced back to the 4th generation. The main points in this genealogical tree were that the patient's father and grandmother were affected with the same condition, also her great-grandfather. Her grandfather and his wife were first cousins.

Ophthalmoscopic Drawings.—Mr. E. Nettleship.

1. A colored drawing of hæmorrhage, probably choroidal in origin, at the macular region in a highly myopic eye, apparently caused by a blow on the corresponding eyebrow. There was also hæmorrhage into the vitreous, and externally some red patches could be seen round the openings for the anterior ciliary arteries.

2. Drawing of a fundus showing scattered spots of posterior choroiditis which are peculiar in being, with one or two exceptions, placed exactly beneath retinal vessels, and these vessels were invariably veins.

3. Drawing of the fundus of a woman aged 45, who had been the subject of syphilis 1½ years previously. The drawing showed active disseminated choroiditis, the special feature being that the choroidal foci were arranged in chains following exactly the course of the retinal veins.

In this connection Mr. Nettleship observed that he had noticed in retinitis pigmentosa, that where the pigmentation surrounded a vessel, this vessel was always found to be a vein and not an artery.

Pituitary Neoplasm with Ocular Symptoms in a Child Aged 10. —Mr. W. Ilbert Hancock.

Emily S., aged 10, first came under observation in May, 1908, with the history of defective sight in the left eye of 14 days' duration. For about a month the child had complained of head-

ache, and the mother had noticed her face swelled at times, more especially on the left side. The general condition was satisfactory except for some carious teeth. R. V. 6/24 and J. 12, the pupil reacted sluggishly to light and the fundus was normal; the L. V. was no P. L., and the pupil was inactive to direct stimulation though acting consensually; the temporal half of the disc was pale. By the end of May, under treatment in the hospital, the vision in the right eye improved to 6/12, and that of the left to finger counting in the temporal field.

In January, 1909, the child again came under observation, when left vision was 6/9 and J. 1, with a pale disc and normal vessels; the field showed temporal hemianopsia. The left eye gave no P. L., with the disc atrophic, though vessels good.

On examining the body generally there was found to be some abnormal adiposity which was definitely greater on the left side than the right. The thryroid and thymus glands were not enlarged; and the nervous system was found normal. The child gives the impression of a stunted, heavily-built, and somewhat overgrown girl.

The discovery of temporal hemianopsia suggested pressure on the chiasma, and an X-ray photograph taken by Mr. Mackenzie Davidson revealed a small roundish body situated in the brain a little in front of the sella turcica about half an inch above the base of the skull and a little to the left of the middle line.

Mr. Davidson subsequently stated that he considered the rounded shadow to be caused by a patch of calcareous deposit in a tumor.

Cyst of the Iris.—Mr. E. W. Brewerton.

M. S., female, aged 28, whose right eye had been excised in 1898 for what was thought to be tubercle, suffered in 1903 from a recurrent attack of plastic iridocyclitis, the pupillary area became blocked with lymph and the lower half of the anterior chamber was filled with organized exudate. The vision had sunk to counting fingers at 10 inches. An optical iridectomy was performed above in 1906 and the vision improved to 5/60. In February, 1907, a small black spot appeared in the lower half of the anterior chamber, the tension was raised, and from this time the eyeball gradually enlarged; the black spot increased in size so that at the present time it measures 10 mm. horizontally and 5 mm. vertically. By transillumination this dark area

is found to transmit light readily, thus excluding new growth; and it was thought that by contraction and retraction of the organized exudate the two pigmentary layers of the iris had become separated and formed a cyst.

Two Cases of Hereditary Optic Atrophy in a Family, with Recovery in One Case.—Dr. Rayner Batten.

The patients were two brothers, aged 24 and 10 respectively, whose sight had diminished to 6/60 in both eyes in each case. The loss of vision was gradual in the elder, having begun at 16, while that of the younger was rapid. The younger patient's sight improved to 6/6 with both eyes in a few months, and the visual field correspondingly enlarged to almost normal, though the discs remained pale. The grandfather of the family was the subject of optic atrophy.

Quinine Amblyopia from a single dose (3i) *of Sulphate of Quinine.*—Mr. Cargill.

E. M., female, aged 28. The sight had failed 9 months previously after taking 3i of quinine sulphate; it was accompanied by severe tinnitus, deafness, and giddiness. The vision gradually improved so that it is now 6/9 in one eye and 6/12 in the other. The irides act to central illumination only, the discs are pale, and the vessels small.

Rodent Ulcer of the Right Eyelid and Nose Treated by X-Rays and Zinc Ionisation.—Mr. Cargill.

G. E., aged 80 years, male, had noticed an ulcer on the right side of the nose for 15 years, and during the last 5 years it had also involved the eyelid. Treatment began in October, 1908, with ionization of the nasal ulcer Zn SO_4 2% using the positive electrode, 2 m.a. being applied for 7 minutes; 2 days later another application was given, and the ulcer healed in 19 days. The ulcer on the eyelid was now being treated with X-rays, and up to the present there had been 20 sittings of seven minutes' duration.

Flattening of Both Corneæ after Phlyctenular Keratitis.—Mr. Hugh Thompson and Mr. F. Hewkley.

The patient, 19½ years old, had suffered from phlyctenular keratitis 6 years ago, from which nebulae had resulted. In 1904

with a +2.5 sphere in both eyes the vision was 6/36; in 1909 it is 6/36 in the right and 6/18 in the left, with +13 D each.

Annular Arcus Senilis in a Man Aged 27.—Mr. Lawson.

This defect had been noticed nine years, and the arcus almost completely surrounded the cornea; both corneæ were faintly nebulous as a result of trachomatous pannus, and it was suggested that there might be some connection between the two conditions.

Optic Atrophy Secondary to Hæmorrhage into Optic Nerve Sheath.—Mr. Lang.

E. J. B., 42, was crushed by a heavy weight (1½ tons) falling on him; the pressure seemed to cause protrusion of the eyeballs, and blood exuded from the nose and mouth. He was blind for 14 days afterwards. At the present time the vision of the right eye is 5/4, field contracted, and lower part of the disc pale, while the left has no perception of light, the disc is atrophic, and the field of vision shows defect in the upper half. The vessels are normal on both sides.

? Lymphosarcoma of the Lids.—Mr. Lang.

This was the case of a man who had been operated on in the Middlesex Hospital for nodular masses of lymphoid tissue growing in both upper lids. The condition had not very much improved, and appeared to be of the nature of a lymphosarcoma.

Hyaline Bodies in the Vitreous.—Mr. Hosford.

A male, aged 73, who had been the subject of syphilis 45 years previously came complaining of failing sight for 6 years. R. V. =6/18, L. V.=6/12. In the right vitreous was a slightly tremulous fibrous diaphragm studded with variously shaped translucent glittering bodies. The left vitreous was also affected.

Retinal Changes the Result of Concussion Injury.—Mr. R. E. Bickerton.

The patient had received a heavy blow on the right side of the face sufficiently severe to crush the zygomatic arch. The right eye became blind immediately afterwards and had not recovered since. The vision was finger counting eccentrically, the pupil reacted to light, and in the macular region there was the appearance of a hole in the retina with a rounded patch of pigmentary disturbance down and out; the disc was pale.

MALCOLM L. HEPBURN.

ABSTRACTS FROM MEDICAL LITERATURE

By J. F. Shoemaker, M.D.,
St. Louis, Mo.

OBSERVATIONS ON CHOKED DISC WITH ESPECIAL REFERENCE TO DECOMPRESSIVE CRANIAL OPERATIONS.

James Borley, Jr., and Harvey Cushing (*Jour. A. M. A.*, Jan. 30) have made a study of about two hundred cases of brain tumor seen in the Johns Hopkins Hospital, and also of as many other cases having choked disc, in such conditions as apoplexy, cerebral thrombosis and embolism, hemorrhage at birth, meningitis, hydrocephalus, nephritis, acute operative compression, post-operative œdema, and the cerebral œdemas which have accompanied cerebral contusion in cases of cranial injury. Their close study of the often rapidly changing appearance of the eye-grounds following decompressive operations, they consider was most instructive. In addition to these studies they have made some experimental investigations in the laboratory which convince them that by mechanical means alone can be produced an acute neuroretinal œdema which will rapidly subside if the pressure is removed in time, but if continued long enough will cause a more permanent swelling of the disc having the histological features which are found in this lesion in clinical cases.

They summarize the views concerning the ætiology of choked disc, give a number of illustrative cases of what decompression does in the way of relieving choked disc and saving vision, and conclude with the following summary:

It is our view that the early injection with stasis of the vessels, the marked œdema with projection of the papilla, and the ultimate round-celled infiltration with new tissue formation which leads to atrophy, are merely stages of the same process. We believe that the mechanical views of Schmidt-Rimpler and Manz are largely correct and that the lesion is primarily due to the crowding into and distention of the sheath of Schwalbe by obstructed cerebrospinal fluid, resulting in an œdema of the nerve-head, in which toxic elements play an insignificant part.

We believe, with Parinaud and his followers, that an acute in-

ternal hydrocephalus in the closed adult skull almost inevitably leads to a choked disc, but we attribute this to the presence of arachnoidal fluid forced into the optic sheath rather than to an œdema spreading from the brain to the optic nerve; furthermore, choked disc is often present with tumors which have not led to hydrocephalus and is usually absent in the "essential" hydrocephalus of infancy.

We believe, since a small, benign, slow-growing and remote tumor so situated as to cause hydrocephalus—for example, by pressure on the iter—can lead to a high grade of choked disc, whereas a malignant, rapidly-growing glioma which causes but little pressure, owing to the way in which it infiltrates the brain, may produce no change whatsoever, even though situated near the optic nerve, that toxic products in the cerebrospinal fluid can hardly be held responsible for any stage of choked disc. With others, we have observed cases of sella turcica tumors which have led to optic atrophy from direct pressure without producing choked disc. In these cases, owing to the situation of the lesion, fluid is not crowded into Schwalbe's sheath.

We believe, since choked disc, contrary to many authorities, is rare in meningitis and equally so in abscess unless there is an obstructive hydrocephalus leading to greatly increased pressure, that meningeal inflammation of itself must be looked on as an unlikely source of the lesion. Furthermore, the process is frequently seen after cranial fractures, apoplexies, and not uncommonly after simple operations when the question of an infection can safely be ruled out.

We believe, finally, since there is an almost uniform subsidence of what we consider to be choked disc (barring certain cases complicated by obstructive hydrocephalus) after decompressive operations, whether conducted for the pressure of tumor, cerebral œdemas of one sort or another (traumatic, nephritic or vascular), infections, or intracranial hæmorrhages, that a mechanical rather than a toxic process must play the chief role in the causation of this well-recognized lesion.

STRABISMUS FROM THE OPERATIVE STANDPOINT.

Peter A. Callan (*Trans. Am. Oph. Soc.*, 1908) notes the fact that ophthalmologists do not agree either as to the method of operation or the time to operate upon a child suffering with strabismus. All agree as to the importance of correcting the

errors of refraction and of orthoptic exercises to train the fusion faculty, but there is a wide diversity of opinion as to when operative interference should be used to straighten the eye if the other means employed do not accomplish this purpose.

Callan believes that we do not operate early enough, and because of this the fusion faculty is lost, never to be regained.

As to the method of operating he thinks that some form of advancement should always be done, employing tenotomy only to increase the effect desired where advancement has not accomplished sufficient. A tenotomy is easily done and often gives good temporary results, but in a young person it frequently produces an over-effect later. Further, it allows the globe to protrude somewhat, and when done on the internus causes sinking of the caruncle. He describes his method of shortening the overstretched muscle, which is a resection.

The advantages of resection over advancement, he states, are as follows: The union of the sutured muscle to the stump is firm and unyielding. There is no danger of an oblique union of the cut surfaces, causing hyperphoria. It is easier of accurate measurement. There is less reaction and after a lapse of a month or six weeks, there is little, if any, trace of the operation.

In resecting the muscle about 1 mm. should be cut away for each five degrees of correction desired on the internus and 1 mm. for each four degrees on the externus. He offers these rules to be followed in the operation of strabismus:

During the growing period of youthful strabotics avoid doing a tenotomy. Operate, however, after six months' use of glasses and orthoptic training show no results.

If the deviation exceeds 30 degrees operate on both eyes with an interval of a year between the two operations, but no tenotomy.

Under 30 degrees confine the operation to the stretched muscles of the defective eye and try for full effect, not under effect.

In the case of adults, where immediate cosmetic effect is only desired, resect the weak muscle and do a slight tenotomy on contracted muscle of same eye at one sitting. Avoid too free dissection of tendon from its capsule. Try for a slight under effect.

The resection should secure the major part of the effect and the tenotomy the minimum. Better still to divide the effect between both eyes, avoiding a tenotomy, if possible.

Granting that in many cases of very high degree of strabismus resection of both eyes may not quite accomplish the desired cosmetic effect, the major part should be due to the resection and only a subordinate role to a slight tenotomy.

A QUERY INTO THE EFFECT OF INTENSE SUNLIGHT ON THE EYE OF THE BLOND TYPE.

E. O. Sisson (*Ophthalmology,* Jan., 1909), in a preliminary paper, offers the following conclusions on this subject:

1. That the normal eyes possess a greater or less amount of pigment, the function of which is to absorb all rays of light not intended to co-operate in producing vision.

2. That, as a rule, the amount of pigment in the human eye corresponds with the general coloring or complexion of the individual. Therefore, the eyes of the brunette type are better protected from the possible harmful effects of intense light.

3. That portion of the human retina, which is the most concerned in vision, namely, the region of the macula, is the best protected by pigment deposits, and this, as we have seen, exists even in the albino, where other portions of the fundus are entirely devoid of pigment.

4. That it is possible that one of the functions of the rhodopsin or visual purple is to protect the delicate nerve tissue of the retina from the harmful effect of light.

5. That pigment does exclude the dangerous actinic rays of light.

6. That the actinic rays are destructive to living protoplasm.

7. That the anatomy and function of any organ of a higher type can be better understood and appreciated by a thorough study of the same organ in a lower type.

8. That the amount of pigment in the eyes of animals and birds and the presence of other structures to protect the eyes from light varies with their habits and the zoological zone they occupy.

9. That owing to the many modifying factors it is very hard to secure clinical data that will prove the theories advanced, but that light does injure the eyes we have ample proof, and it is possible that some of the eye diseases whose ætiology is at present obscure, may find their explanation in this way.

10. That vernal conjunctivitis or spring catarrh is conceded to be a disease dependent more or less upon the harmful effects of solar light.

11. That in view of the fact that glass stops the ultra-violet light, while quartz allows it to pass through, we have in the wearing of glasses a protection from the harmful rays.

12. That all of the above facts show the great necessity for pigments in the iris, retina and chorioid and that the density of the pigment must be in proportion to the degree of light to which that type of man is exposed.

REVIEWS.

REFRACTION AND HOW TO REFRACT, including sections on optics, retinoscopy, the fitting of spectacles and eyeglasses, etc. By J. Thorington, A.M., M.D. Fourth Edition. 220 illustrations, 13 of which are colored. Philadelphia: P. Blakiston's Son & Co. 1909. Price $1.50.

As with the previous editions, it gives us great pleasure to recommend to the ophthalmic student this carefully worked out manual. The only thing we would like to see changed, is the use of the verb *to refract.*

MANUEL PRATIQUE DE STRABISME. (Practical handbook on strabismus.) By Dr. R. Onfray; with a preface by Dr. Rochon-Duvigneaud. Paris: G. Steinheil. 1909.

In the preface to this book Dr. Rochon-Duvigneaud states that the French language hitherto possessed no book which could serve as a complete guide for the clinical and therapeutic study of strabismus. The book, therefore, is intended to fill a necessary gap in the French language. We have no doubt it will do so, as it is a very complete and well written treatise on strabismus in all its forms and the different methods of treatment, preceded by chapters on the physiology of binocular vision.

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